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TITLE: PIEZOELECTRIC ELEMENT FOR INJECTOR

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ABSTRACT:

PROBLEM TO BE SOLVED: To provide a piezoelectric element usable for a long time and having excellent durability when used in an injector.

SOLUTION: The piezoelectric element 1 is built into the injector and generates driving force for the injector. When a coercive electric field of the piezoelectric element 1 is Ec with a preset load of 500 N applied to the piezoelectric element, d(0.1 Ec)/d(1.2 Ec) > 0.43 holds as a relationship between an apparent piezoelectric constant d(1.2 Ec) calculated from a static elongation obtained when an electric field of 1.2 Ec is applied in the same

direction as a direction of polarization and an apparent piezoelectric constant d(0.1 Ec) calculated from a static elongation obtained when an electric field of 0.1 Ec is applied in the same direction as the direction of polarization.

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DETAILED DESCRIPTION

[Detailed Description of the Invention]

[0001]

[Field of the Invention] this invention relates to the laminating type piezo-electric-crystal element used as a driving source of an injector.

[0002]

[Description of the Prior Art] Injectors (fuel injection equipment), such as an internal combustion engine of an automobile, change the pressure state which changes the switching condition of a fuel path and is given to a nozzle needle by ***** which moves the valve element of the method valve of three connected to the common rail which accumulated for example, high-pressure fuel, or the method valve of two, and by changing a nozzle needle into a valve-opening state, they are constituted so that fuel may be injected.

[0003] And generally as a driving source which moves the above-mentioned valve element, the solenoid valve etc. is used. On the other hand, for the purpose of controlling the above-mentioned driving source finely and performing precise control of a fuel-injection state, as shown in JP,11-229993,A, the attempt which is going to use a laminating type piezo-electric-crystal element as the above-mentioned driving source has been made.

[0004]

[Problem(s) to be Solved] However, although the injector which used the piezo-electric-crystal element for the driving source is proposed like the above, it has not yet resulted in utilization. In an injector, it is necessary to repeat spraying of fuel very much at high speed. For example, 10,000 spraying or more may be performed in 1 minute. Therefore, a very severe busy condition is imposed on the piezo-electric-crystal element used as this driving source. And in the conventional piezo-electric-crystal element, what can fully bear the above severe busy conditions was not yet developed, without causing a crack etc. [0005] this invention was made in view of this conventional trouble, and when it applies to an injector, it tends to offer the piezo-electric-crystal element which was excellent in usable endurance over the long time.

[0006]

[Means for Solving the Problem] Invention of a claim 1 is a piezo-electric-crystal element which generates the driving force of this injector while being built in an injector, this piezo-electric-crystal element It comes by turns to carry out the laminating of two or more piezo-electric layers and the internal-electrode layer for applied-voltage supply which are displaced according to applied voltage, and this piezo-electric-crystal element When the anti-electric field are set to Ec, where a 500-N presetting load is given to the above-mentioned piezo-electric-crystal element The piezoelectric constant d of the appearance computed from the static elongation when giving the electric field of 1.2Ec(s) in the same direction as the direction of polarization (1.2Ec) It is in the piezo-electric-crystal element for injectors characterized by materializing d (0.1Ec) / relation of d(1.2Ec) > 0.43 between the piezoelectric constants d of the appearance computed from the static elongation when giving the electric field of 0.1Ec(s) in the same direction as the direction of polarization (0.1Ec).

[0007] The point which should be most noted in this invention is that d (1.2Ec) is size from d (0.1Ec)/0.43. the piezo-electricity which begins a variation rate almost immediately by the voltage seal of approval in case a piezo-electric-crystal element displaces -- a variation rate -- a component and 90-

degree rotation component which begins a variation rate with after [a voltage seal of approval] delay -- existing -- these sum totals -- the whole variation rate -- it becomes an amount

[0008] the variation rate at the time of this invention persons giving 1.2 times as many electric field as the anti-electric field Ec (it mentions later for details) further -- an amount -- the above-mentioned piezo-electricity -- a variation rate -- the variation rate at the time of being the thing of the sum total with a component and 90-degree rotation component, and giving 0.1 times as many electric field as the anti-electric field Ec -- an amount -- 90-degree rotation component -- almost -- there is nothing -- almost -- piezo-electricity -- a variation rate -- the variation rate only by the component

[0009] therefore, the piezo-electricity which will contribute to the variation rate in the above-mentioned piezo-electric-crystal element if those ratios are computed by asking for these apparent piezoelectric constants d, respectively -- a variation rate -- the rate of an abundance ratio of a component can be obtained namely, the piezo-electricity at the time of a piezo-electric-crystal element displacing Above d (0.1Ec)/d (1.2Ec) -- a variation rate -- it becomes the value which substituted for the rate of an abundance ratio of a component

[0010] Here, let the value of Above d (0.1Ec)/d (1.2Ec) be a larger value than 0.43 in this invention. thereby -- a variation rate -- the inside of a component, and piezo-electricity -- a variation rate -- a piezo-electric-crystal element with the rate of an abundance ratio of a component higher than 90-degree rotation component can be obtained And in this case, since there are few ratios of 90-degree rotation component, calorific value accompanying the repeat of the variation rate of a piezo-electric-crystal element can be lessened. Moreover, so, the endurance of a piezo-electric-crystal element can be raised. [0011] Therefore, according to this invention, when it applies to an injector, the piezo-electric-crystal element which was excellent in usable endurance over the long time can be offered.

[0012] Moreover, it is more desirable like invention of a claim 2 that d (0.1Ec) / relation of d (1.2Ec) >=0.5 is materialized between the above-mentioned piezoelectric constant d (1.2Ec) and the above-mentioned piezoelectric constant d (0.1Ec). Thereby, the above-mentioned operation effect can be demonstrated still more certainly.

[0013] Invention of a claim 3 is a piezo-electric-crystal element which generates the driving force of this injector while being built in an injector. next, this piezo-electric-crystal element It comes by turns to carry out the laminating of two or more piezo-electric layers and the internal-electrode layer for applied-voltage supply which are displaced according to applied voltage, and this piezo-electric-crystal element [when alternating voltage is impressed so that 1.5kV //mm / field strength may arise from 0 by the sin wave, where a 500-N presetting load is given] The rate of change of the amount of displacement at the time of changing the frequency of the voltage to impress from 1Hz to 200Hz is in the piezo-electric-crystal element for injectors characterized by being smallness from 9%.

[0014] The point which should be most noted in this invention is that the rate of change of the amount of displacement in the above-mentioned conditions is smallness from 9%. When this rate of change is 9% or more, drive speed of a piezo-electric-crystal element can seldom be raised. In addition, as small, since the more nearly high-speed drive of this rate of change is attained, it is more desirable. So, 7% or less is more desirable, the above -- a variation rate -- a variation rate in case the frequency of the voltage which the rate of change of an amount impresses is 1Hz -- an amount -- the variation rate in Y1,200Hz -- when an amount is set to Y200, it is the value expressed by 100x (Y1-Y200) / Y1 Moreover, let the amount of displacement be a value 5 seconds after after voltage impression.

[0015] Next, it explains per operation effect of this invention. the piezo-electric-crystal element of this invention -- the above -- a variation rate -- the rate of change of an amount is smaller than 9% That is, even if it raises the frequency to impress, the amount of displacement seldom falls. And even if it raises frequency and raises drive speed, sufficient amount of displacement is obtained. Therefore, the piezo-electric-crystal element of this invention is stabilized, and has drive speed raised. And even if the number of times of injection for 1 minute is 10,000 times, the variation rate (expansion and contraction) of a piezo-electric-crystal element can be repeated with a margin. So, the piezo-electric-crystal element of this invention demonstrates the endurance which was excellent when it applied to an injector, and is usable over a long time.

[0016] Invention of a claim 4 is a piezo-electric-crystal element which generates the driving force of this injector while being built in an injector next, this piezo-electric-crystal element It is in the piezo-

electric-crystal element for injectors which comes by turns to carry out the laminating of two or more piezo-electric layers and the internal-electrode layer for applied-voltage supply which are displaced according to applied voltage, and is characterized by the amount of displacement increasing by the rise of temperature within the limits of -40 degrees C - 150 degrees C, as for this piezo-electric-crystal element.

[0017] The point which should be most noted in this invention is that the amount of displacement in the above-mentioned specific temperature requirement increases with the rise of temperature.

[0018] Next, it explains per operation effect of this invention. In the injector using the piezo-electric-crystal element, since the displacement loss by the viscosity of fuel falling and leaker increasing and the displacement loss by the bulk-modulus fall of fuel arise, in connection with a temperature rise, the amount of required displacement of a piezo-electric-crystal element increases.

[0019] To change of this amount of required displacement, the control circuit of an amendment sake is needed in this. However, in order to perform the amendment by the circuit, enlargement of a control circuit scale will be caused. On the other hand, the piezo-electric-crystal element of this invention has the property of increasing the amount of displacement in connection with a temperature rise. Therefore, the control circuit which controls the amount of displacement can be made into comparatively easy structure, and the miniaturization can be attained. So, it becomes easy to apply the piezo-electric-crystal element of this invention to an injector.

[0020] Moreover, as for the rate of increase of the amount of displacement in a -40 degrees C - 150 degrees C temperature requirement, it is desirable like invention of a claim 5 that it is 5 - 40%. In this case, increase of the amount of required displacement of the piezo-electric-crystal element accompanying the above-mentioned temperature rise is easily suppliable, the above -- a variation rate -- the rate of change of an amount -- the variation rate in -40 degrees C -- an amount -- the variation rate in Y-40,150 degrees C -- when an amount is set to Y150, it is the value expressed by 100x (Y150-Y-40) / Y-40

[0021] Invention of a claim 6 is a piezo-electric-crystal element which generates the driving force of this injector while being built in an injector. next, this piezo-electric-crystal element It comes by turns to carry out the laminating of two or more piezo-electric layers and the internal-electrode layer for applied-voltage supply which are displaced according to applied voltage, and is in the piezo-electric-crystal element for injectors to which dielectric loss which asked for this piezo-electric-crystal element from the P-E hysteresis is characterized by being smallness from 8%.

[0022] The point which should be most noted in this invention is that the dielectric loss for which it asked from the above-mentioned P-E hysteresis is smallness from 8%. In the graph which took Charge P for the field strength E which carried out the seal of approval to the horizontal axis along the vertical axis, the above-mentioned P-E hysteresis takes tracing of the value of the charge P at the time of making it back-fall which raised the above-mentioned field strength to 1.5kV/mm, and is acquired (refer to the example of an operation gestalt).

[0023] When the dielectric loss for which it asked from this P-E hysteresis is 8% or more, exoergic temperature becomes high and there is a problem of seldom raising drive speed. Therefore, considering as 7% or less is more desirable. In addition, as the dielectric loss for which it asked from this P-E hysteresis is small, it is more desirable from generation of heat being suppressed.

[0024] Next, it explains per operation effect of this invention. The dielectric loss which asked for the piezo-electric-crystal element of this invention from the P-E hysteresis like the above is smallness from 8%. Therefore, as shown also in the example of an operation gestalt mentioned later, when a piezo-electric-crystal element is driven at high speed, generation of heat of a piezo-electric-crystal element can be suppressed, and endurance can be raised remarkably. So, the piezo-electric-crystal element of this invention demonstrates the endurance which was excellent when it applied to an injector, and is usable over a long time.

[0025]

[Embodiments of the Invention] It explains using <u>drawing 1</u> - <u>drawing 5</u> about the piezo-electric-crystal element for injectors concerning the example of an operation form of example of operation form 1 this invention. The piezo-electric-crystal element 1 for injectors of this example is a piezo-electric-crystal element which generates the driving force of an injector 5 while being built in an injector 5, as shown in

drawing 5. This piezo-electric-crystal element 1 comes by turns to carry out the laminating of two or more piezo-electric layers 11 and the internal-electrode layers 21 and 22 for applied-voltage supply which are displaced according to applied voltage, as shown in drawing 1. And the piezo-electric-crystal element 1 is in the state which gave the 500-N presetting load to the piezo-electric-crystal element 1. when the anti-electric field are set to Ec. The piezoelectric constant d of the appearance computed from the static elongation when giving the electric field of 1.2Ec(s) in the same direction as the direction of polarization (1.2Ec) Between the piezoelectric constants d of the appearance computed from the static elongation when giving the electric field of 0.1Ec(s) in the same direction as the direction of polarization (0.1Ec), d (0.1Ec) / relation of d(1.2Ec) > 0.43 is materialized. Hereafter, this is explained in full detail. [0026] As are shown in drawing 1 and drawing 2, and the piezo-electric-crystal element 1 serves as positive/negative by turns in the above-mentioned internal-electrode layers 21 and 22 between the layers of the above-mentioned piezo-electric layer 11, it forms and it becomes. As shown in this drawing, one internal-electrode layer 21 is arranged so that it may expose to one side 101, and the internal-electrode layer 22 of another side is arranged so that it may expose to the side 102 of another side. And the side electrodes 31 and 32 were formed so that the side 101,102 of the piezo-electric-crystal element 1 might be made to flow through the edge of the exposed internal-electrode layers 21 and 22.

[0027] Moreover, in the piezo-electric-crystal element 1, the portion arranged in a part for the center section of the direction of a laminating so that a mechanical component 111 and this may be pinched was made into the buffer section 112, and the portion arranged so that this buffer section 112 may be pinched further was made into the dummy section 113.

[0028] The manufacture method of this piezo-electric-crystal element 1 and detailed structure are explained. The piezo-electric-crystal element 1 of this example can be manufactured using the green-sheet method used widely. Weighing capacity of the green sheet is carried out so that it may become composition of a request of powder, such as a lead oxide which serves as the main raw material of piezoelectric material by the well-known method, a zirconium oxide, titanium oxide, a niobium oxide, and a strontium carbonate. It was made for final composition to serve as the so-called PZT (PZT) in this example. moreover, leaden evaporation -- taking into consideration -- the above -- a mixing ratio -- it prepares so that it may become rich 1 to 2% rather than the stoichiometry of composition d (0.1Ec)/d (1.2Ec)0.43 [in addition,] which tuned the content of the composition component of PZT finely and was mentioned above in this example -- size -- 0.5 or more and the bird clapper were aimed at preferably And these raw materials are blended dryly with a mixer, and temporary quenching is carried out at 800-950 degrees C after that.

[0029] In addition, from the above d (0.1Ec)/d (1.2Ec)0.43, as size and electrostrictive ceramics which becomes 0.5 or more preferably, although the composition component of Above PZT was adjusted, it can also obtain by adjusting the composition component using others and various electrostrictive ceramics.

[0030] Subsequently, pure water and a dispersant are added to temporary-quenching powder, it considers as a slurry, and wet grinding is carried out with a pearl mill. A solvent, a binder, a plasticizer, a dispersant, etc. are added and this trituration object is mixed with a ball mill, after carrying out powder degreasing, dryness and. Then, vacuum degassing and viscosity control are carried out, stirring this slurry with an agitator within vacuum devices.

[0031] Subsequently, a slurry is fabricated to the green sheet of fixed thickness with doctor blade equipment. The collected green sheet is pierced with a press machine, or a cutting machine cuts it, and it is fabricated on the rectangle object of a predetermined size. The green sheet is common to a mechanical component, the buffer section, and the dummy section.

[0032] Subsequently, for example, silver/palladium = screen-stencil fabrication of the pattern is carried out on one front face of the green sheet after fabrication with the paste (henceforth a Ag/Pd paste) of the silver and palladium which consist of 7/3 of ratios. An example of the green sheet after pattern printing is shown in drawing 3 (a). In addition, the same sign is substantially given to the same portion on account of explanation.

[0033] In the front face of a green sheet 11 used as a piezo-electric layer, all over [this] abbreviation, mist and the small pattern 21 (22) are formed and it considers as the internal-electrode layer 21 (22) with the above-mentioned Ag/Pd paste. The agenesis section 119 of the internal-electrode layer 21 (22) is

formed in one opposite side side of the front face of a green sheet 11. That is, this has been arranged so that the internal-electrode layer 21 (22) may not arrive at one edge (portion equivalent to the side 101 or ** 102 of the piezo-electric-crystal element 1) of the opposite side of a green sheet 11 but the internal-electrode layer 21 (22) may reach the other-end section which counters.

[0034] The green sheet 11 in which such an internal-electrode layer 21 (22) was formed is prepared by predetermined laminating number of sheets based on the requirement specification of a mechanical component 111 and the amount of buffer section 112 displacement. Moreover, the green sheet 12 which is not printing the internal-electrode layer the buffer section 112 and for dummy section 113 also makes required number-of-sheets preparations.

[0035] Subsequently, these green sheets 11 and 12 are piled up. <u>Drawing 4</u> shows the laminating state of green sheets 11 and 12, and serves as an exploded view of the piezo-electric-crystal element 1 substantially. In piling up the green sheet 11 in which the internal-electrode layer 21 (22) was formed, it piles up so that the agenesis section 119 of an electrode may be located in the left-hand side in drawing, and right-hand side by turns. The internal-electrode layer 21 attained and exposed to the side 101 on the right-hand side of [in drawing] a green sheet 11 serves as an internal electrode of one pole by this, and the internal-electrode layer 22 attained and exposed to the side 102 of the left-hand side in drawing serves as an internal electrode of the pole of another side.

[0036] And in the central mechanical component 111, as shown in <u>drawing 4</u>, a laminating is carried out only using the green sheet 11 in which the above-mentioned internal-electrode layer 21 (22) was formed, the green sheet 12 which does not form the internal-electrode layer between green sheets 11 in the buffer section 112 is made to intervene, a laminating is carried out, and a laminating is carried out only using the green sheet 12 which does not form the internal-electrode layer in the dummy section 113. This becomes the layered product of the structure shown drawing 2.

[0037] Subsequently, it degreases by the 400-700-degree C basis with an electric furnace after the thermocompression bonding by the warm water rubber press etc., and calcinates by the 900-1200-degree C basis. Subsequently, the external electrodes 31 and 32 are formed by applying and printing the above-mentioned Ag/Pd paste or Ag paste on the side of the above-mentioned layered product. The external electrode 31 is formed in the position which the internal-electrode layer 21 of one pole has exposed, and takes the flow of each internal-electrode layer 22. The external electrode 32 of another side is formed in the position which the internal-electrode layer 22 of the pole of another side has exposed, and takes the flow of each internal-electrode layer 22. Then, this is immersed into insulating oil, direct current voltage is impressed between the internal-electrode layer 21 and 22 from the external electrodes 31 and 32, the piezo-electric layer 11 is polarized, and the piezo-electric-crystal element 1 is obtained.

[0038] In addition, like the above, by using the green sheet (piezo-electric layer) 12 of the same quality

of the material as the piezo-electric layer 11 used for the mechanical component 111, as the kind of the above-mentioned dummy section 113 of manufacture material did not increase, it aimed at reduction of a manufacturing cost. However, the piezo-electric layer 12 of this dummy section 113 may be constituted from an another material, for example, an insulating magnetic material can also constitute. [0039] It sets for the piezo-electric-crystal element 1 of this example here, an important point When the anti-electric field are set to Ec, where a 500-N presetting load is given to the piezo-electric-crystal element 1. The piezoelectric constant d of the appearance computed from the static elongation when giving the electric field of 1.2Ec(s) in the same direction as the direction of polarization (1.2Ec). It is the point that d (0.1Ec) / relation of d(1.2Ec) > 0.43 is materialized between the piezoelectric constants d of the appearance computed from the static elongation when giving the electric field of 0.1Ec(s) in the same direction as the direction of polarization (0.1Ec).

[0040] Here, the anti-electric field Ec are explained first. <u>Drawing 6</u> is explanatory drawing of the above-mentioned anti-electric field Ec. the field strength (voltage) which gives this drawing to a horizontal axis at a piezo-electric-crystal element -- a vertical axis -- a variation rate -- an amount is taken In addition, field strength considers the same side as the direction of polarization as plus (+), and considers the direction of polarization, and an opposite side as minus (-).

[0041] And it starts from A points, a field strength seal of approval is first carried out to the piezo-electric-crystal element 1 in the same direction with the direction of polarization, and the value is raised gradually. According to this, the amount of displacement of the piezo-electric-crystal element 1

increases. Next, after field strength reaches the B point which is 150V, field strength is gradually made low. According to the fall of this field strength, the amount of displacement decreases shortly. And after field strength is set to 0, field strength is continuously reduced in the direction contrary to the direction of polarization gradually. According to this, the amount of displacement decreases further. In this example, when field strength is set to -90V (C points), the amount of mutation starts to increase. The absolute values (90V) of the field strength of this point are the anti-electric field Ec in this invention. [0042] And after field strength reaches after that D points which are -150V, field strength is raised again. According to this, the amount of displacement decreases shortly. And if field strength is set to 0 and raises field strength in the direction of polarization further, the amount of mutation will start to increase. Although this field strength of D points is also called anti-electric field, Ec as used in the field of this invention does not carry out. In this invention, the anti-electric field at the time of carrying out the seal of approval of the voltage in the direction contrary to the direction of polarization to the last are set to Ec. By raising field strength further after that, finally it will be in the almost same state as the B point, and the same behavior will be repeated after that.

[0043] It asks for each above-mentioned piezoelectric constants d (0.1Ec) and d (1.2Ec) based on calculated Ec like the above. It can ask for a piezoelectric constant with the inclination of the graph at the time of creating the rate of change of the variation rate at the time of making the field strength which carries out a seal of approval gradually increase, i.e., the graph of the relation between field strength and the amount of displacement. Therefore, in this example, the displacement-voltage hysteresis in the case of making the seal of approval of the field strength of 150V carry out in the same direction as the direction of polarization was searched for.

[0044] <u>Drawing 7</u> takes field strength (voltage) (V) along a horizontal axis, and takes the amount (micrometer) of displacement along a vertical axis. And where a 500-N presetting load is given from the upper and lower sides of the piezo-electric-crystal element 1, the seal of approval of the voltage is carried out in the same direction as the direction of polarization, and the value is increased gradually. And tracing of the amount of displacement is plotted to <u>drawing 7</u>. In this example, it carried out until field strength was set to 150V. In addition, although it plotted when reducing field strength in this drawing until it was gradually set to 0 from 150V, and it asked for the hysteresis curve, only the case where field strength is made to increase in this example is important.

[0045] And each above-mentioned piezoelectric constant can be defined by rate of change in case the value is 0.1Ec(s) (9V), and the rate of change in the case of being 1.2Ec (108V), when making field strength increase. In this example, the piezoelectric constant d (0.1Ec) was 0.37, and the piezoelectric constant d (1.2Ec) was 0.68. Therefore, these ratios were obtained by 0.37/0.68, are 0.54 and had fully exceeded 0.43.

[0046] Next, an example of an injector which can use the piezo-electric-crystal element 1 of the above-mentioned composition as a driving source is explained briefly. An injector 5 is applied to the common rail injection system of a diesel power plant, as shown in <u>drawing 5</u>. This injector 5 has the up housing 52 with which the above-mentioned piezo-electric-crystal element 1 as a mechanical component is held, and the lower housing 53 with which it is fixed to the soffit and the injection nozzle section 54 is formed in the interior, as shown in this drawing.

[0047] It is pillar-shaped and, as for the up housing 52, insertion fixation of the piezo-electric-crystal element 1 is carried out into the approximate circle dugout 521 which carries out eccentricity to a medial axis. The high-pressure fuel path 522 is established in the side of a dugout 521 in parallel, and the upper-limit section is open for free passage to the external common rail (****) through the inside of the fuel introduction pipe 523 which projects in the up housing 52 top section.

[0048] The fuel which the fuel delivery tube 525 which is open for free passage to the drain path 524 protrudes on the up housing 52 top section, and flows out of the fuel delivery tube 525 is returned to a fuel tank (****). The drain path 524 opens for free passage and uses the inside of the vertical housing 52 and 53 as the method valve 551 of three later mentioned by the path which is prolonged caudad, and which is not illustrated from this crevice 50 further via the crevice 50 between a dugout 521 and a mechanical component (piezo-electric-crystal element) 1, and is **.

[0049] The injection nozzle section 54 is equipped with the nozzle hole 543 which injects the high-pressure fuel which is opened and closed by the nozzle needle 541 which slides on the inside of the

ID NO: 3, wherein a difference of a cytosine to thymine substitution at nucleotide position 172 of the SorCS1 cDNA sequence indicates that the human is a candidate for developing type 2 diabetes.

The nature of the invention, therefore, requires the knowledge of predictive associations between position 52 relative to SEQ ID NO: 4 or position 172, relative to SEQ ID NO: 3 or the "SorCS1" cDNA, and susceptibility to developing type 2 diabetes.

The claims recite "protein coding region" or "cDNA" of the SorCS1 gene, however it is known that in mice, different isoforms of SorCS1 exist. The specification does not teach the different isoforms of human SorCS1. Further, the designation of specific position 172 with reference to SEQ ID NO: 3 is unclear because position 172 of SEQ ID NO: 3 is a g, not a cytosine (or thymine). The specification does not teach any other SorCS1 cDNA sequence in humans.

The invention is in a class of inventions which the CAFC has characterized as 'the unpredictable arts such as chemistry and biology" (Mycolgen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Federal Circuit 2001)).

The specification teaches that the inventors began by narrowing the genetic region associated with severe type 2 diabetes to a 7 MB segment of mouse chromosome 19 (page 4, para 0017). The specification teaches that 2 genes previously found in the region were SorCS1 and SorCS3, which belong to a family sharing a large region of similarity including the VPS10 domain. The specification teaches that due to similarity with sortilin, SorCS1 and SorCS3 are expected to be involved in insulin-stimulated glucose transportation and in controlling body fat metabolism. The specification teaches that the 7MB region was characterized and that it was

found that the only difference between severely diabetic mice and less severely affected mice was 3 mutations in SorCS1, leading to 3 amino acid changes (table 1). The specification, however, does not teach the specific function or activity of SorCS1. The specification does not teach if other mutations occurred in other portions of the mouse genome that may be responsible for the severe form of diabetes observed in the mice.

The specification provides no teaching or working examples of any mutations in any portion of the SorCS1 gene in humans, or an association between SorCS1 alleles in a human subject and type II diabetes susceptibility. The specification asserts at page 3 that the SorCS1 gene in mice is "directly analogous" to the human gene, however this statement is unclear. The genes are not identical, and the meaning of "directly analogous" cannot be determined. For example, at table 1, the specification teaches different mutations at specific positions of mouse SorCS1. The specification teaches a mutation; at position 1140 from Ser to Phe, and at position 1150 from Ser to Pro. However, in humans position 1140 is Asp, and position 1150 (in SEQ ID NOS 4) is His. None of these amino acids are "directly analogous" to either amino acid found in mice at each position. Although, the specification has been amended to recite a mutation at position 52 from Thr to Ile (also found in SEQ ID NO: 4), the specification provides no teaching of the specific function or activity for SorCS1, or any of these 3 positions, accordingly the affect of each amino acid at such positions is unpredictable. Additionally, claims 1 and 3 broadly encompass any nucleotide change which leads to an isoleucine substitution at position 52, however only a single specific nucleotide substitution which leads to this alteration in mice is taught. The specification does not teach if the amino acid substitution is the causative mutation which leads to a more severe form of diabetes in mice, or if the nucleotide change may be in

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linkage with another allele. Therefore, given the lack of guidance from the specification as to any mutations in any region of the SorCS1 gene in humans, a teaching of the function of SorCS1 including critical amino acids and domains required for function, or a predictable correlation between the presence of SorCS1 mutations and diabetes susceptibility in other species, the skilled artisan would be unable to predict an association between the claimed positions in the protein coding region or cDNA of the SorCS1 gene in humans and susceptibility to type 2 diabetes.

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The specification's assertions with regard to putative SorCS1 activity is based on homology analysis with sortilin and the family of proteins that contains a VPS10 domain (page 4, end of para 00017). However, it is known for nucleic acids as well as proteins that even a single nucleotide or amino acid change or mutation can destroy or alter the function of a biomolecule in many instances, albeit not in all cases. The effect of these changes are largely unpredictable as to which ones have a significant effect verses not. The prior art does not teach the function of SorCS1 or how it is involved in type 2 diabetes. The post filing specifically date art provides some characterization of SorCS1 (see Hermey et al, JBC, vol. 278, Feb. 2003, pages 7390-7396), but teaches that neither the mature luminal domain nor any of the cytoplasmic domains of the different SorCS1 isoforms bound any of the ligands previously shown to interact with sortilin and SorLA, demonstrating sorCS1 is functionally different from the previously characterized Vps10-D family receptors (para bridging pages 7390-7391). Additionally, Hermey teaches that the different isoforms of SorCS1 have completely different cytoplasmic domains that mediate different trafficking in cells (abstract). It is clear that the art supports that SorCS1 has a different function than other Vps10 domain family members, and that the 3 different

isoforms of SorCS1 do not function in the same manner where the different cytoplasmic domain for each isoform mediates different trafficking in cells.

Claims 1-3 and 12 appear to be drafted based on the amended specification's recitation that a difference was found in the SorCS1 gene between B6 and BTBR mice corresponding to amino acid position 52. The specification asserts "It appears that the activity of the SorCS1 protein may determine islet mass. Alternatively, the SorCS1 protein may affect insulin secretion in pancreatic beta cells or insulin degradation in the kidney or liver" (page 8, para 00033), however the specification does not teach the function of SorCS1, or whether or how the change from Thr to Isoleucine altered the function or activity of the SorCS1 nucleic acid or protein such that the change provides an increased susceptibility to type 2 diabetes in mice. Accordingly, the affect of the mutation of Thr to Ile, is unpredictable in humans. The specification provides no guidance as to whether this mutation, or the other mutations listed in Table 1, occurs in a critical region or domain or how it affects the function or activity of a critical region or domain, such that the skilled artisan would be able to predict the same effects in humans. The specification provides no teaching or working examples that this mutation, or the other mutations listed in Table 1, exists in humans or would have a similar affect in humans. Kahn teaches that disruption of a specific gene in mouse models of diabetes does not necessarily provide a predictable correlation that any polymorphism in the corresponding human sequence would be similarly associated (Kahn, Cell, vol. 92, pages 593-596, 1998, cited in the IDS). Kahn teaches "Withers et al., reported that disruption of IRS-2 causes diabetes in mice. The most compelling aspect of this report is that inactivation of this single gene causes defects in both insulin action and insulin secretion." (page 593, last para of col. 2). However, Kahn further teaches "The parallels between the IRS-2 knockout mice and Type 2 diabetes in humans raises the tantalizing question as to whether human diabetes is

caused by mutations in the IRS-2 gene. Disappointingly, studies in press in several populations, including Danish Caucasians... reveal no association between polymorphisms in the IRS-2 gene and Type 2 diabetes" (page 594, 2nd full para in col. 2).

The instant specification provides no teaching or guidance as to the role of critical amino acids in any of the isoforms of either murine or human SorCS1 nor how such are involved in susceptibility to type 2 diabetes. The specification provides no predictable association that any alteration, in any protein coding region or cDNA of the SorCS1 gene, including those claimed, in humans, let alone any species, is diagnostic or indicates a susceptibility for developing type 2 diabetes. No predictable correlation between the structural alterations in the mouse sequence and susceptibility for developing type 2 diabetes has been taught by the specification. The specification does not teach the function of SorCS1 nor how it's function, or lack of function, or altered function are predictably associated with type 2 diabetes.

The quantity of experimentation in this area is extremely large as it requires analysis of claimed positions in the SorCS1 gene to determine whether the isoleucine variant at position 52 of SEQ ID NO: 4, or the nucleotide variant T, at position 172 of SEQ ID NO: 3, which would alter the Gly amino acid at codon 55 to valine, is associated with type 2 diabetes. As neither the art nor the specification provide guidance as to whether the amino acids at such positions are critical to the function of SorCS1 or are in some way associated with diabetes susceptibility such analysis is replete with trial and error experimentation, with the outcome being unpredictable.

In order to practice the invention as claimed, one would first have to establish that a predictive relationship exists between the disclosed mutations and susceptibility to type 2 diabetes in humans. Such experimentation could involve functional analysis of a protein whose

actual function has to be determined as well. The experimentation could also involve a large study of patients and controls to screen for mutations in SorCS1 in humans to determine whether the claimed mutations are associated with susceptibility to type 2 diabetes in humans. Such analysis represents an inventive and unpredictable undertaking with each of the many intervening steps not providing any guarantee of success.

Thus, given the broad claims in an art whose nature is identified as unpredictable, the state of the prior art, the lack of guidance in the specification, the breadth of the claims and the quantity of experimentation necessary to practice the claimed invention, it would require undue experimentation to practice the invention commensurate in scope with the claims.

Response to Arguments

The response traverses the rejection. The response asserts that the specification sets forth at page 5, para 00019 that the genomic and cDNA sequences of mouse SorCS1 were known to those of skill in the art at the time of filing. This argument has been thoroughly reviewed but was not found persuasive as this statement could not be found in the paragraph indicated. The response further asserts that the degree of identity between the mouse and human SorCS1 coding region is sufficient to soundly predict that applicant's genetic evidence from the congenic mouse model is predictive of the same genetic phenomenon in humans and that the term 'directly analogous' is intended to mean that the mouse and human SorCS1 genes are similar in structure and function. At page 10, the response summarizes the teachings of the specification and concludes that applicants discovered the difference in susceptibility to diabetes resolved down to differences in the alleles of the gene for SorCS1, and that since the same correlation exists in mice also exists

in humans and since the corresponding homologous SorCS1 gene having a conserved threonine at amino acid position 52 is found in both mice and humans, the same susceptibility to developing type 2 diabetes is found in humans and in mouse. This argument has been thoroughly reviewed but was not found persuasive. Although figure 2 provides an alignment between the three isoforms in mice as well as human SorCS1, this alignment provides no analysis regarding regions or domains which are critical to activity or function. The genetic background of the congenic mice and humans would not be expected to be the same. It is not clear, from the teachings in the specification, whether the mutations disclosed in the specification are the cause for the difference in diabetes susceptibility between the B6 and BTBR mice, or whether one or more of the mutations act in concert with or are linked to the causative mutation which could be hundreds or thousands of nucleotides away in a different gene or on a different chromosome. It is not clear from the teachings of the specification, or the assertions in the response, that the only difference in the mice genetically, was that leading to the 3 amino acid differences in table 1, or that the Thr to Ile mutation occurred at position 52. It is additionally unclear, and the specification does not teach whether the Thr to Ile change provided for a difference in activity or function of SorCS1, to establish a predictive association between this amino acid change and T2D in any genetic background. The specification provides no correlation between the structure of the specific mutation in Table 1 and their affect on the function or activity of SorCS1 to provide for T2D susceptibility. With regard to the assertion that "since the same correlation that exists in mice also exists in humans, and since the analogous SorCS1 gene having a conserved Threonine at amino acid residue 52 is found in mouse and humans, the same susceptibility to developing T2D should be found in humans and in mouse", it

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is noted that the meaning of "the same correlation exists in mice also exists in humans" is unclear, because it is not clear which "correlation" is being referred to. The response at page 11, also asserts that "among the genes analyzed in mice, SorCS1 is the only gene which applicants detected ... expression level differences. In fact, applicants believe that these expression differences cause the increased diabetes risk". These arguments have been thoroughly reviewed but were found unpersuasive. The specification does not appear to disclose any expression levels differences detected for SorCS1, nor does it provide any guidance that the mutations disclosed in table 1 led to expression level changes. Accordingly, these arguments are not persuasive. Additionally, such argument is considered Attorney's arguments and cannot take the place of evidence on the record. As stated in the MPEP, 2106 "Arguments of Counsel"

"However, it must be emphasized that arguments of counsel alone cannot take the place of evidence in the record once an examiner has advanced a reasonable basis for questioning the disclosure. See In re Budnick, 537 F.2d at 538, 190 USPQ at 424; In re Schulze, 346 F.2d 600, 145 USPQ 716 (CCPA 1965); In re Cole, 326 F.2d 769, 140 USPQ 230 (CCPA 1964). For example, in a case where the record consisted substantially of arguments and opinions of applicant's attorney, the court indicated that factual affidavits could have provided important evidence on the issue of enablement."

With regard to SorCS1 function, at page 9, the response reiterates the teachings of para 00033 of the specification, and asserts "Specifically, it is believed the reduced or altered insulin levels in the congenic mice are a result of a decreased insulin secretion in vivo, which is associated with disrupted islet morphology. Furthermore, it is noted that the cellular function of SorCS1 is still unknown, however it is known to bind platelet growth factor-BB. This growth factor is required for the recruitment of pericytes or their precursor to vascular endothelial cells, where they stabilize the microvasculature and have a key role in blood vessel development. Maintenance of proper islet vasculature is important for both insulin secretion and islet survival and thus may be of particular relevance to the mammalian phenotype disclosed in the instant

specification." This argument has been thoroughly reviewed but was unpersuasive to overcome the rejection. First, the instant specification has provided no teaching of the specific function of SorCS1 other than speculation on it's activity with regard to islet mass or alternatively it's affect on insulin secretion or insulin degradation (para 00033). None of the mutations disclosed are discussed with regard to such function, nor is any guidance provided by the specification on the affect of such mutations with regard to the possible functions disclosed in the specification and asserted to in the response. Second, it is further noted that the specification does not teach these assertions made in the response. These assertions are considered attorney's arguments and cannot take the place of evidence on the record.

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Written Description

10. Amended claims 1-3 and newly added claims 12 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed. had possession of the claimed invention.

Amended claim 1 is drawn to a method of assessing whether a human subject is susceptible to type 2 diabetes by determining the SorCS1 cDNA sequence of that subject, deducing the amino acid sequence encoded thereby, and comparing it with SEQ ID NO: 4, wherein a difference of a threonine to isoleucine substitution at amino acid position 52 of the SorCS1 amino acid sequence indicates that the subject is susceptible to developing type 2 diabetes. Amended claim 2, is drawn to a method of assessing whether a human subject is susceptible to type 2 diabetes by

determining the cDNA sequence of the subject in the SorCS1 gene and comparing it with SEQ ID NO: 3, wherein a difference of a cytosine to thymine substitution at nucleotide position 172 of the SorCS1 cDNA sequence indicates that the subject is susceptible to developing type 2 diabetes. Amended claim 3 is drawn to a method of determining if a human being is a candidate for developing type 2 diabetes by determining the sequence of the protein coding region of the human SorCS1 gene in the genome of the human, deducing the amino acid sequence encoded by the region sequenced, and comparing the deduced amino acid sequence to SEQ ID NO: 4, wherein a difference of a threonine to isoleucine substitution at amino acid position 52 of the SorCS1 amino acid sequence indicates that the human being is a candidate for developing type 2 diabetes. Newly added claim 12 is drawn to a method of determining whether a human subject is a candidate for developing type 2 diabetes by determining the cDNA sequence of the subject in the SorCS1 gene and comparing it with SEQ ID NO: 3, wherein a difference of a cytosine to thymine substitution at nucleotide position 172 of the SorCS1 cDNA sequence indicates that the human is a candidate for developing type 2 diabetes.

The amended claims are drawn to "Assessing whether a human subject is susceptible to type 2 diabetes comprising the step of determining the protein coding region [cDNA sequence] of the human SorcS1 gene" wherein comparison to SEQ ID NO: 4 or 3 respectively and a difference at amino acid 52 or the nucleic acid 172, respectively, indicates that the human subject is a candidate for type 2 diabetes. However, the specification provides no basis for these specific nucleotide or amino acid positions in humans.

At para 00020 of the specification, the specification generally sets forth diagnostic use for examining humans for their SorCS1 gene and determining differences with respect to SEQ ID

NO: 4. Although the specification recites specific positions in Table 1, these positions are with regard to differences found in B6 vs BTBR mice, both of which, were diabetic, albeit with differing severity. However, the specification does not appear to set forth diagnostic methods for diabetes susceptibility in humans by determining any *particular* mutation or position.

Accordingly, the newly added claims directed to diagnostic methods in humans at a specific SorCS1 position appears to introduce new matter into the instantly claimed invention.

Additionally, as the mutations disclosed were found in mice not in humans, there is no evidence that such mutations would even exist in any human SorCS1 nucleic acid or protein sequence. The designation of specific position 172 with reference to SEQ ID NO: 3 is further unclear. Position 172 of SEQ ID NO: 3 is a guanine, not a cytosine (or thymine). The specification does not teach any other SorCS1 cDNA sequence, let alone any sequences with mutations, in humans.

With regard to the recitation of amino acid "52", in table 1, at page 4, the specification was amended to change the amino acid position from "50" to "52" for Thr/Ile, B6/BTBR respectively. However, the specification does not appear to provide support for this specific change. At page 12, first paragraph, the response asserts that this change was made to correct an "inadvertent misnumbering". However, as set forth in the MPEP 2163 (I) (B): "While there is no in haec verba requirement, newly added claim limitations must be supported in the specification through express, implicit, or inherent disclosure. An amendment to correct an obvious error does not constitute new matter where one skilled in the art would not only recognize the existence of the error in the specification, but also recognize the appropriate correction. In re Oda, 443 F.2d 1200, 170 USPQ 268 (CCPA 1971)." In the instant case, the specification does not appear to

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provide guidance that the correct amino acid position is 52. While a threonine is present in the hSorCS1 amino acid sequence at position 52, threonine is also present at, for example, amino acid 68, as well as in the different mouse SorCS1 isoforms (see Figure 2). Given the limited guidance in the specification, it does not appear that one of skill in the art would not have recognized the existence of the error or the appropriate correction. The arguments with regard to the specification objection are not persuasive for the reasons made of record above.

Conclusion

- 11. No claims are allowed.
- 12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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Jehanne Sitton

Primary Examiner

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7/23/07